



August 19-22, 2018
Montpellier, France



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21/08/18

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Variations of genes copy number in populations of Bluetongue-virus

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AIMS:

Viruses have the largest diversity of genome architectures. Among them, segmented viruses possess a genome divided in several chromosomes or segments, encapsidated together. The most prevalent hypothesis to explain the evolution of this genome organisation is an increased ability for genetic exchanges. Recently a hypothesis has suggested that this genome organisation could have evolved to favour the regulation of gene copy number and thus gene expression at the population level. In an infected host, a viral population is composed of different genotypes, including defective genotypes lacking specific segments. Under this new hypothesis, the segment copy number in the population differs among segments, and consequently among the genes they bear. This situation would lead to a differential expression of viral genes at the host level, influencing phenotype. Our study aims to test whether variations in segment copy number exist in populations of Bluetongue virus, whose genome harbour a segment number among the highest described. Bluetongue virus follows a complex life cycle involving compulsory alternation between insect vectors and ruminants. This virus could thus use different segment copy numbers to differentially regulate gene expression in its highly unrelated hosts. To test our hypothesis, we designed a QPCR assay for each of the 10 genomic segments in order to monitor quantitative variations in different hosts. We first isolated a BTV genotype (BTV4 serotype) from sheep infected in Corsica in 2017. This isolate was used to infect two different cell lines, a mammalian line (VERO) and an insect cell line (KC). Gene copy number variation in segmented genome virus could be a key mechanism underlying their high adaptive capacity and supporting an alternative way of adaptation to host alternation. This study is a prelude to Bluetongue virus gene copy number variation monitoring with time, space and environmental variations.